ence in the cells of large quantities of a double light refracting fat which characterizes the cells of the cortical adrenal cells. This constituent may be found in the cells of even the most remote metastasis, though in the case which we report it has seemed to have disappeared in the brain metastasis.

In addition to this histological resemblance to the adrenal gland the location of the tumor is also most significant. Besides its occurrence in the adrenal gland itself, where normally we find small adenomas of minute size, hypernephromas occur most commonly in the upper pole of the kidney and in the right lobe of the liver on its under surface. These parts are in very close proximity in the embryo, and when we find metastasis also occurring in the ovaries and testicles, organs which take their origin in the same close proximity, though later descending to their respective sites, we can hardly doubt the origin of this tumor. This conception of hypernephromas must lead one to regard it as a congenital phenomenon. Later in life, sometimes, we see it take on an activity signifying an entire change of character. It starts to grow rapidly, to invade surrounding tissues and organs, and to disseminate metastases in many different parts of the body. The most common place to find these secondary tumors is in bone, but we here record a metastasis in the brain, in which location many statistics regarding hypernephromas record only one or two instances.

METABOLIC OBSERVATIONS ON AMYOTONIA CONGENITA.

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Amyotonia congenita, or myatonia congenita, is a rare condition affecting children, aged up to three or four years, and exceptionally later, with a general proximal pseudoparalysis, for which no satisfactory etiology or pathology has been advanced. According to a recent clinical review of the subject by Haberman,[1] about 35 cases had been reported up to 1910. Since then a case has occurred in the service of Dr. J. P. Crozer Griffith at the University Hospital, Philadelphia, and was reported by him.[2] Griffith has collected 49 cases, including Haberman's and his own, although he admits that the diagnosis of some of them is doubtful. According to either series, however, the rarity of the disease is evident. Griffith's case, a boy, aged fifteen months, on admission, December, 1910, showed the characteristic findings of amyotonia congenita —extreme muscular weakness without signs of muscular atrophy and without evidence of rickets. The general nutrition appeared to be good. There was absence of the tendon reflexes, and entire absence of contraction both to faradic and galvanic currents.

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[1] Amer. Jour. Med. Sci., 1910, cxxxix, 383.

[2] Archiv f. Kinderheilkunde, 1910, liv, 211; Trans. Amer. Ped. Soc., 1910, xxii, 184.

It occurred to us that some metabolic studies in the case would be of interest. The child's condition during the first few months of his stay in the hospital precluded the possibility of making metabolic observations owing to his extreme asthenia which was increased, from time to time, by slight febrile attacks, the exact cause for which could not be determined. In June, 1911, when he was aged twenty-one months, his condition having improved, we were able, through the courtesy of Dr. Griffith, to obtain a five-day observation. The publication of our findings has been postponed until they could be controlled by similar studies upon normal children of the same age, which we have now made.

In October, 1911, the child developed an attack of bronchitis, quickly followed by pneumonia, which resulted fatally within five days. The autopsy was performed by Dr. B. S. Veeder— the results of which, together with the histological study, were published by Drs. Griffith and Spiller.[3] A summary of their important findings in the muscular and nervous system showed a brain of extraordinarily large size (as large as that of an adult), but otherwise presenting no gross anomaly. The nerve cells of the anterior lobes were scarce, and those present were much atrophied. The anterior roots throughout the cord were smaller than normal, and stained imperfectly by the Weigert-hematoxylin method. A peripheral nerve examined was found to be much degenerated. The intramuscular nerve fibers also showed degeneration. The muscular system (back, forearm, and calf) showed marked atrophy of the fibers in some places; in others the fibers were well preserved. There was an excess of fibrous and fatty connective tissue, increase in size of some muscle fibers, and fatty change within others. The muscle spindles appeared to be normal. From their study of this case and a review of others reported with autopsy, Griffith and Spiller conclude that in the lighter cases of amyotonia the pathological changes may be confined to the muscular tissues. In intense cases the nervous system is affected in a marked degree. The relations of the disease to muscular dystrophy and to the Werdnig-Hoffmann type of muscular atrophy are as yet undetermined. The points of resemblance to both these diseases cannot be ignored.

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[3] Amer. Jour. Med. Sci., 1911, cxlii, 165.

There is grouped under the caption of neurology, however, a considerable variety of diseases, for whose inclusion there little or no etiological or pathological justification can be advanced. Some of these diseases to be sure, do not belong clearly in any other branch of medicine; but in any event they have been largely removed from the consideration of workers in lines outside those of neuropathology, in the broader sense of the word. In the absence of means of attack other than histological study little or no progress has been made toward their solution. Examples of this are well seen in the condition now under discussion, in the muscular dystrophies, paralysis agitans, myasthenia gravis, etc.

In certain previous communications,[4] one of the present writers has endeavored to approach some of these conditions from a different standpoint embodying a "dynamic point of view;" that is to say, a consideration of the body metabolism. The results were interesting and seemed to justify further attempts of the sort when opportunity presented. But few such studies are on record.

There seems to be no adequate reason for the indifference with which these interesting diseases have been regarded in this country by internists and laboratory men at large since the inclusion of such conditions within the realm of neurology is, largely, the result of excursions by neurologists into the field of medicine.

The phenomena of diseases such as those mentioned above are obviously of the widest importance in studying the metabolism of the muscles and of the body as a whole, as is so well exemplified in our recently acquired knowledge of tetany, for instance. But one observation seems to be on record, however, of the metabolic features in amyotonia congenita, and that one is by Spriggs, who studied the uric acid and creatinin and found a low output of the latter in a boy, aged four and one-half years (0.06 gram creatinin *per diem*).

The experiences above referred to in regard to Myasthenia gravis and Myotonia atrophica developed an interesting departure from the normal in the former disease which suggested the advisability of including a consideration of the calcium metabolism in further studies on allied conditions. The case to be reported was therefore, studied with respect to the urinary nitrogen, fecal nitrogen, urinary calcium, fecal calcium, and the creatinin. For control the metabolism of 2 normal children of the same age was also observed. All 3 children were placed upon the same diet and were kept in Bradford frames to facilitate collections. The difficulties incidental to metabolic studies upon young children are too well known to require mention here except to say that they operated to prevent our obtaining an accurate balance in the case of amyotonia. The essential facts sought for were elicited, however, in regard to the creatinin and fairly clearly indicated in regard to the calcium.

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[4] Pemberton, Amer. Jour. Med. Sci., June, 1910; ibid., February, 1911.

Although an accurate balance was impossible in the case of amyotonia, it was obtained in the 2 control cases as a further index of comparison and for possible future reference.

CASE I.—John, aged thirty-three months; normal; control case.

AVERAGE DIET.

Breakfast:	Bread	24 grams
	Butter	7 grams
	Eggs	51 grams
	Milk	100 c.c.
Dinner:	Rice	43 grams
	Butter	7 grams
	Crackers	13 grams
	Milk	100 c.c.
Supper:	Bread	36 grams
	Butter	5 grams
	Eggs	48 grams
	Milk	100 c.c.

Ingesta.

Nitrogen	12.4721 grams
Calcium	1.4604 grams

CASE II.—Alvin, aged forty months; normal; control case.

AVERAGE DIET.

Breakfast:	Bread	36 grams
	Butter	10 grams
	Eggs	54 grams
	Milk	100 c.c.
Dinner:	Rice	38 grams
	Bread	19 grams
	Butter	14 grams
	Milk	100 c.c.
Supper:	Rice	57 grams
	Crackers	13 grams
	Butter	10 grams
	Milk	100 c.c.

Ingesta.

Nitrogen	11.9805 grams
Calcium	1.4248 grams

CASE III.—Charles, aged twenty-one months; amyotonia congenita.

Diet.

	Nitrogen.	Calcium.
Bread	6.8816 grams	0.1594 grams
Rice	0.8103 grams	0.0752 grams
Egg	4.7388 grams	0.2055 grams
Butter	0.1048 grams	0.0372 grams
Crackers	0.7548 grams	0.0139 grams
Milk	16.4479 grams	3.7652 grams
	29.7382	4.2564

	Before metabolic period.	After metabolic period.	Average.	Kgs.
Weight, Case I, John	24.5 oz.	24.14 oz.	24.9 oz.	11.16
Weight, Case II, Alvin	24.4 oz.	24.8 oz.	24.6 oz.	11.07
Weight, Case III, Chas.	21.8 oz.	22.8 oz.	22.0 lbs.	10.00

Case I.—John. April, 1912.

	7th	8th	9th	10th	11th
Quantity of urine	257 c.c.	L	320 c.c.	175 c.c.	155 c.c.
Nitrogen of urine	3.7900 grams	O	4.0592 grams	3.3712 grams	2.4416 grams
Creatinin of urine	0.1041 grams	S	0.1149 grams	0.0547 grams	0.0662 grams
Calcium of urine	Not done	T	0.1168 grams	0.0507 grams	0.0528 grams

Total nitrogen in total feces = 1.7112 grams. Total urinary nitrogen = 10.4720 grams
Total calcium in total feces = 1.7054 grams. Total urinary calcium = 0.2203 grams

Total nitrogen egested	12.1832 grams
Total calcium egested	1.9257 grams

Positive nitrogen balance +0.2889 grams. Weighed 24 lbs., 5 oz. before metabolic period
Negative calcium balance —0.4653 grams. Weighed 24 lbs., 14 oz. after metabolic period.
Daily creatinin average, 0.0786 grams. Creatinin coefficient, 7.04 mg.

Case II.—Alvin. April, 1912.

	10th	11th	12th
Quantity of urine	154 c.c.	238 c.c.	298 c.c.
Nitrogen of urine	2.1905 grams	4.4382 grams	3.1752 grams
Creatinin of urine	0.0738 grams	0.1083 grams	0.0850 grams
Calcium of urine	0.1615 grams	0.2651 grams	0.1554 grams

Total nitrogen in total feces = 1.4483 grams. Total urinary nitrogen = 9.8039 grams.
Total calcium in total feces = 1.2640 grams. Total urinary calcium = 0.5829 grams.

Total nitrogen egested	11.2522 grams
Total calcium egested	1.8469 grams

Positive nitrogen balance = +0.7283 grams
Negative calcium balance = —0.4221 grams
Daily creatinin average = 0.08903 grams
Creatinin coefficient = 7.04 mg.

CASE III.—Charles. June, 1910.

	10th	11th	12th	13th	14th
Quantity of urine	226 c.c.	168 c.c.	213 c.c.	199 c.c.	218 c.c
Nitrogen of urine	4.627 gms.	3.957 gms.[5]	3.288 gms.	3.7419 gms.	3.864 gms.[7]
Creatinin of urine	0.017 gms.	0.015 gms.	0.018 gms.	Too low to be read[6]	Too low to be read[6]
Calcium of urine .	0.07677 gms.	0.02624 gms.[5]	0.0136 gms.	0.00889 gms.	0.0057 gms.
			No stool today		0.87192 gms. Ammonia nitrogen.

Total nitrogen of feces . .	2.2862 grams	Total nitrogen of urine . .	19.478 grams
Total calcium of feces . .	2.6971 grams	Total calcium of urine . .	0.1312 grams

Total nitrogen egested	21.7642 grams
Total calcium egested	2.8283 grams

Ingested nitrogen,	29.7382 grams	gained 1 pound	Positive nitrogen balance
Egested nitrogen	21.7642 grams		
	7.9740 grams		7.9740 grams
Ingested calcium,	4.2564 grams		Positive calcium balance.
Egested calcium,	2.8283 grams		
	1.4281 grams		1.4281 grams

Daily creatinin average	0.0166 grams
Creatinin coefficient	1.66 mg.
Daily creatinin average in Sprigg's case	0.0625 grams
Creatinin coefficient in Sprigg's case	4.0 mg.
Normal, 7 to 11 mg.	

(Creatinin coefficient = the daily creatinin elimination in milligrams of creatinin per kilo of body weight.)

A consideration of the figures reveals some points of interest. The creatinin shows a great departure in Case III (amyotonia) from both of the normal controls and even from Sprigg's case. The average daily creatinin output, when it could be estimated at all, was 0.0166, whereas in Sprigg's case it was 0.0625, and in our 2 normal cases, 0.0786 and 0.0890 respectively. This gave creatinin coefficients as follows:

Amyotonia congenita (Case III)	=0.66 m. creatinin
Amyotonia congenita (Sprigg's case)	=4.0 m. creatinin
Normal child, Case I	=7.04 m. creatinin
Normal child, Case II	=8.04 m. creatinin

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[5] These figures are missing and are averaged from those preceding and following. Some urine was spilled by an attendant, leaving enough for creatinin only, though the total quantity was known from the ward record. Having all the calcium figures but those for one day, it is possible to approximate a balance, since the urinary calcium is low in any event. The nitrogen balance can be approximated in the same way, though with greater possible error.

[6] No reading possible with 10, 20, or even 40 c.c. urine. This was corroborated by Dr. Wm. Welker.

[7] On this day the nitrogen of the ammonia was approximately 22.3 per cent. of the total nitrogen.

Until we know the full meaning of the creatinin excretion, interpretation of such results must be guarded, but they would seem to be valuable, at least from the diagnostic standpoint, in differentiating amyotonia from the many conditions with which it may be confused, especially if the involvement be slight. To what extent these figures, together with Sprigg's, indicate a true disturbance of the internal muscular metabolism, is problematical but they are certainly highly suggestive.

As mentioned before, an exact calcium balance was impossible in our case, though in any event the inequalities in the excretion of calcium necessitate guarded conclusions as to its elimination, unless the metabolic period be protracted or the differences observed great. The development of fever after the last metabolic day recorded, prevented the continuation of our observations and subsequently the condition of the child, never very robust, precluded a repetition of the experiment. So far as our figures go, however, and the general facts seem fairly clearly indicated, there is no evidence of marked disturbance in the calcium metabolism.

In observations on a case of myasthenia, previously mentioned, a decided calcium loss was noted over a metabolic period of a week, and it was thought that the present case might show some analogy to it; the opposite is suggested, however.

In conclusion, obligation should be expressed to the Board of Managers of the Presbyterian Hospital, Philadelphia, for the installation of the facilities which have made this and other studies possible. Acknowledgment is also due Dr. Damon B. Pfeiffer, director of the pathological laboratory, for his courteous coöperation on many occasions.